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FREE COMMUNICATIONS XVII

Coagulation: Clinical.

HIGH GENE FREQUENCY OF FACTOR XI DEFICIENCY IN ASHKENAZI JEWS. Uri Seligsohn.
Institute of Hematology, Chaim Sheba Medical Center, Tel Hashomer, Israel.

Factor XI (fXI) deficiency has previously been reported mainly in Jews. Its frequency and ethnic distribution however, have not been determined. It was the purpose of this study to examine these questions.

Since 1966, 78 patients with fXI deficiency have been observed. They belong to 36 unrelated families, all of whom are of Eastern European origin (Ashkenazim).

Of 428 normal Ashkenazi Jewish subjects whose fXI levels were assayed, 1 subject had severe deficiency (3%), 35 partial deficiency (24 - 49%) and 392 had normal levels. The 95% confidence limits of the frequency of severe fXI deficient subjects in the total Ashkenazi Jewish population are 0.1 - 0.3%, and for partial deficient subjects 5.5 - 11%.

Since severe as well as partial fXI deficient patients may not be diagnosed until profuse bleeding is presented following trauma, the observed high gene frequency warrants performing the appropriate tests in all Ashkenazi Jewish patients undergoing surgery.

INHIBITION OF FACTOR XI BY MYELOMA PROTEIN M. A. Rimon, I. Raz and M. Lahav. Tel-Aviv University, Tel Aviv, Israel.

Waldenstrom's macroglobulinemia sometimes involves hemostatic disorders. It has been assumed that this may be due to interaction of the myeloma protein with some blood coagulation factor(s). In the present investigation the sera of seven myeloma patients were studied for a possible interaction between the myeloma protein and clotting factors II, VIII, IX and XI.

Normal human plasma was incubated for 30 min at 37°C with different dilutions of patients' serum or of purified myeloma proteins. The ability of this mixture to restore the clotting times of various deficient plasmas was tested by the partial thromboplastin time test.

Factor XI activity of normal plasma was substantially inhibited upon incubation with patient's serum or with purified myeloma protein M. This effect seems to be specific for myeloma protein M since it was neither observed with myeloma proteins G or A, nor with normal IgM. Other coagulation factors investigated so far were not inhibited by myeloma protein M. It is concluded that this protein is specifically affine to factor XI resulting in hindering the latter's activity.